Lacunar Stroke

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Case Presentation

A 37-year-old, right-handed, monogamous homosexual white man was transferred to the Massachusetts General Hospital in December 1984, following admission to another hospital 8 days previously. On the day of his initial admission he had noticed the sudden onset of left-sided facial numbness and, within seconds, numbness of the left arm and leg leading to difficulty in controlling his leg movements with walking. An electroencephalogram, echocardiogram, and computed tomographic (CT) scan performed shortly after admission were normal. Within 12 hours the left-sided facial numbness resolved, by 36 hours the left arm was no longer numb, and by 72 hours the sensation in the left leg was normal. During the following days he noted the waxing and waning of a "coldness" of the left arm.

There was no history of headache, nausea, vomiting, or weakness of the left hand with the present illness. The patient reported dizziness with a vertiginous sensation on following finger movements.

The patient's past history was notable for severe headaches associated with vomiting since the age of 5 years, but he had never previously experienced weakness or numbness. In 1982 he began to notice an unusual difficulty in controlling movements of the left leg. Shortly thereafter he experienced severe lower back pain after an episode of sneezing, which gradually dissipated. However, his gait deteriorated dramatically at that time. In the past several years he had also noted a clumsiness of his left hand when playing the piano and occasional episodes of "jumbled vision" at the end of the day.

Hypertension was first noted during a hospitalization in January 1984 for right-sided ureteric colic. The blood pressure ranged from 150/105 to 135/95 mm Hg during this admission and was still elevated (136-140/94-104 mm Hg) when he was seen as an outpatient 3 months later. Hydrochlorothiazide, 50 mg q.i.d., was commenced, and the pressure fell to 116 to 124/80 to 84 mm Hg at subsequent outpatient visits. The ureteric colic resolved with spontaneous passage of a calculus. He had had two previous episodes of right-sided ureteric colic, one necessitating nephrolithotomy in 1976. The stone removed at that time was composed of calcium oxalate. Investigations during the January 1984 admission included a renogram (performed after passage of the calculus), which was normal apart from the suggestion of renal cysts. An abdominal ultrasound was performed, which showed only two cysts at the upper pole of the right kidney.

Investigations relating to his hypertension following the January 1984 admission excluded secondary causes of hypertension, and an electrocardiogram was normal, as were the results of biochemistry tests, blood count, and urinalysis.

Examination at the time of his December 1984 admission revealed a blood pressure of 136/96 mm Hg. The results of the remainder of his cardiovascular ex-
amination were normal, and no arrhythmias or bruits were noted. Central nervous system examination showed the pupils to be equal and reactive. The disks were slightly pale, but no retinopathy was present. Eye movements were full, and facial movements were symmetrical. The results of the remainder of the central nervous system examination, including cranial nerves, motor, sensory, parietal, cerebellar function and reflexes, were normal.

Results of relevant investigations were as follows. Visual evoked responses and brainstem auditory evoked responses were normal. Somatosensory evoked responses from the left hand (median nerve) showed an abnormally decreased amplitude of the N19 waveform, consistent with a definite, persistent abnormality in sensory conduction above the lower medulla. Results of a lumbar puncture, including a Venereal Disease Research Laboratories (VDRL) test and an immunoelectrophoretogram, were normal. Angiography, including visualization of the left vertebral artery, right internal carotid artery, and right external carotid artery, failed to reveal any abnormalities. The blood count, electrolyte levels, erythrocyte sedimentation rate, liver and renal function, and blood lipid levels were normal. Cultures of blood, urine, and cerebrospinal fluid were sterile. Hepatitis B surface antigen and antibody were not detected, the rheumatoid factor was less than 60 IU/ml, and a test for antinuclear antibody was positive at 1:16 (homogeneous pattern). Levels of CH50, C3, and C4 were all normal. An electrocardiogram showed normal sinus rhythm. A nuclear magnetic resonance imaging (MRI) study of the central nervous system showed a small, right-sided thalamic infarct.

Five days after admission he experienced a further short-lived episode of left-sided facial numbness, but he remained well thereafter. Propranolol, 20 mg q.i.d., was commenced, which further controlled the blood pressure to diastolic levels below 90 mm Hg. He was discharged 8 days after admission and has remained well during a follow-up period of 8 months.

Case Discussion

The recognition of lacunar syndromes has been both challenging clinically and relevant therapeutically. Initially, they were considered to be the result of a unique arteriopathy encountered in hypertension. The clinical syndromes seemed relatively pure since they occurred mainly in the motor pathways or in the thalamus and produced pure motor or pure sensory stroke. Clinical recognition was important, since the arteriopathy affected vessels too small to be seen on angiography and affected too little of the brain substance to disturb the electroencephalogram. Because the arteriopathy was the same as that in hypertensive parenchymatous hemorrhage, anticoagulation was considered too risky to warrant casual use.

From this pristine beginning, the subject has expanded in many directions. Numerous clinical studies have shown that the syndromes may result from nonvascular diseases. Many of these reports can be criticized for glossing over important differences between these instances of nonvascular disease and those with autopsy-proven lacunes. Once CT scanning technology developed to the point that small lesions could be imaged easily, the earlier insistence on autopsy studies was all but forgotten. Although many authors still attempt to explain the lesion as ischemic, some exercise no such caution. As a result the term lacune is now so widely used for any small, deep lesion that it has lost much of its value.

General Features

Lacunes are most often encountered in a setting of hypertension and are estimated to account for as many as 15% of strokes. Their size ranges from 0.2 mm to as large as 15 mm. Almost all occur in the territories of the lenticulostriate and thalamoperforant arteries and in the paramedian branches of the basilar artery. They are not thought to result from a "small vessel disease" scattered throughout the brain, although casual use of the term often conveys this notion. Lacunes are rare in the surface gray matter, major white matter of the cerebral hemispheres, visual radiations, corpus callosum, medulla, and spinal cord, despite the presence in such locations of vessels of similar small size. Interest among neuroradiologists in Binswanger dementia has even resurrected earlier notions of an arterial disease of the long arteries penetrating into the cerebrum.

The lenticulostriates, thalamoperforants, and paramedian basilar branches, which are all less than 500 μm in size, arise directly from the larger 6- to 8-mm internal carotid or basilar artery. Their small size and point of origin rather proximally in the arterial network are thought to expose these vessels to forces that scarcely reach other similar-sized arteries in the cerebral cortex. Their lack of collaterals produces infarction in a cylindrical or cone shape distally from the point of arterial occlusion to the end of the arterial territory.

A surprisingly leisurely mode of onset characterizes many lacunar strokes. In contrast to embolism, in which a gradual onset is encountered in less than 5% of cases, up to 30% of lacunes develop over a period of up to 36 hours. During this time a mild weakness may evolve to total paralysis, usually by intensifying the initial deficit but occasionally by spreading into limbs not affected initially. This smooth onset occurs with equal frequency in all types of lacunar syndromes. The sudden onset typical of other ischemic strokes occurs in only 40% of cases. The rate of evolution of the stroke does not appear to predict the severity of the eventual defect.

Vascular Pathology

Mural deposits of fatty macrophages and fibrinoid material in the penetrating arteries are the usual cause of lacunes. There has been no end of theories about the pathophysiology of the arterial disease. The literature
devoted to such theories can be ordered along a spectrum of the effects of severe, moderate, and mild hypertension.

Fibrinoid necrosis is said to occur in severe hypertension, affecting arterioles and capillaries of the brain, retina, and kidneys. It appears histopathologically as a brightly eosinophilic, finely granular, or homogeneous deposit involving the connective tissue of blood vessels. High pressure, found only with such extreme blood pressure elevation as occurs in hypertensive encephalopathy and eclampsia, possibly aggravated by the constricted arteries, produces increased capillary hydrostatic pressure and capillary damage. Overdistention of these small arteries occurs in segmental fashion and leads to vascular necrosis, which allows red blood cells, plasma, and protein ultrafiltrates into the stretched segments of the wall. For reasons not easily explained, larger vessels are not as greatly affected. Perhaps they absorb enough of the pressure wave in the subintima and in their thicker muscularis to resist distention to degrees sufficient to produce such change, while the arteries of the same small size in the cerebrum may be protected by their more distal location.

Lipohyalinosis and fibroid necrosis are thought to be similar processes, sharing some of the same histoch- emical, electron microscopic, and immunofluorescent characteristics. However, the two conditions differ histochemically in that fibrinoid necrosis is reported to stain strongly for phosphotungstic acid–hematoxylin whereas lipohyalinosis does not. Lipohyalinosis is found most commonly in a setting of chronic, nonmalignant hypertension. It is thought to be an intermediate stage between the fibrinoid necrosis of severe hypertension and the microatheroma associated with more long-standing hypertension.

Whether such efforts to separate the two conditions are warranted by the data is debatable. Some authorities consider them identical. Little guidance can be found in the literature, where what seems to be identical disorders have been labeled as lipohyalinosis, hyalinoses, hyaline fatty change, hyaline arterionecrosis, angionecrosis, plasmatic vascular destruction, atherosclerosis of small arteries, segmental arterial disorganization, and fibrinoid arteritis.

It is disappointing to note how few detailed studies of brain lacunes have been recorded. The biggest single contributor has been C.M. Fisher. Much of his most recent work has brought up the frequency of microatheroma as a common cause of lacunes, perhaps responsible for the larger, symptomatic lacunes. To my knowledge, his is the only study of a consecutive series of symptomatic lacunes.

In passing, mention should be made of Charcot-Bouchard aneurysms. Although such lesions are commonly said to be the cause of lacunes, direct evidence of a connection is meager and no recent evidence has appeared to support the suggestion that lipohyalinosis is the end-stage of an earlier Charcot-Bouchard aneurysm.

A few other causes of deep infarcts are worth noting. Atherosclerosis of a major cerebral or basilar artery may affect a penetrating artery whose mouth originates at the site of an atheromatous lesion. This mechanism should be expected to produce the larger lacunes, since the occlusion would result in infarction of the entire territory of the penetrating artery instead of just a portion of it. Microembolism has been inferred as a cause in a few severely sectioned lacunes shown to have normal arteries leading to the infarct. Macroembolism was probably the cause of the infarct in Patient 10 among the original descriptions of pure motor stroke by Fisher and Curry. Even polycythemia has been thought a cause of lacunes, the small vessels being obstructed by the sludged blood. Dissection of a tiny artery may occur in the process leading to Charcot-Bouchard aneurysms.

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Varying forms of arteritis may also occur, especially due to chronic meningitis (so-called Heubner's arteritis) in chronic neurosyphilis, any severe granulomatous meningitis, and chronic fibrosing meningitis. Arteritis may have been a major cause of small, deep infarcts when chronic neurosyphilis was in its heyday; however, two major works on the subject contain no specific instances, although the authors opined that "they undoubtedly occur." This opinion was not shared by Pentschew, who expressed doubts whether "syphilitic endarteritis" was actually of syphilitic origin.

### Laboratory Studies

As a diagnostic tool, CT scanning has been a bit disappointing. Even the most modern CT scanners may miss lacunes smaller than 2 mm in the internal capsule. Thalamic lacunes are usually even smaller and are routinely missed. In addition, CT scan artifacts are so common in the brainstem that only the largest lacunes are imaged. The size of the abnormality on CT scan is misleading: autopsy correlations show that the CT scan overestimates the size of the lacune by as much as 100%. Scanning within 10 days of onset detects as many as 55% of lacunes that eventually show on CT scan. The larger the lacune observed on CT scan, the more disease involving the parent major cerebral artery itself should be suspected.

Use of MRI is still in its infancy. Work from this institution has indicated that MRI may show a brainstem abnormality missed by CT scan and may better explain the syndrome than does the corona radiata lesion imaged by computed tomography. The pure sensory or sensorimotor syndrome described in the present patient proved difficult to image by either technique. The bigger the motor deficit, the more likely the CT scan will be abnormal. In pure sensory stroke, the MRI (but not the CT) scan may be abnormal when the entire half of the body is involved, but partial sensory syndromes usually have a normal MRI as well as CT scan.

Evoked cerebral responses have shown alterations in the sensory evoked response waveform that suggest a subclinical sensory impairment in clinically pure motor strokes. Efforts to find an abnormality in the sensory evoked potential were disappointing in a personal...
series; only those patients with a large lesion on CT scan and an accompanying motor deficit showed such abnormalities.

The electroencephalogram is usually normal, probably because of the small size of the lesion. Clinical Types

Pure motor stroke is the most frequently encountered clinical type of lacune, occurring as often as 60% in a prospective series. Cases have been reported from focal infarction involving the corona radiata, internal capsule, pons, and medullary pyramid. Associated syndromes include ataxic hemiparesis and the dysarthria–clumsy hand syndrome. Movement disorders have occurred from infarcts of lacunar size in the head of the caudate nucleus and adjacent corona radiata, the subthalamic nucleus, and the thalamus.

Pure sensory stroke is the syndrome closest to that seen in the present patient. It occurs less frequently than does pure motor stroke and is attributed to a thalamic lacune. Observations on the arterial disease are confined to two patients. In one, a microtheroma was found. Whether the lacune was symptomatic was unmentioned. In the other, a pure sensory syndrome was described clinically. The 54-year-old patient was recovering from a right-sided pure motor hemiplegia when a feeling of "pins and needles" developed in the left lower lip, left side of the mouth, and the fingers of the left hand; the sole of the left foot tingled and felt numb, dull, and swollen many hours later. No sensory deficit was evident on examination. Unpleasant paresthesias affected the left side of the face and the left foot. A CT scan was normal on the fourth day. At autopsy 6 months later, a 2 X 2 X 3.7-mm lacune was found in the right ventral posterior nucleus, which was fed by four tiny arteries that arose from a single artery Destroyed by lipohyalinosis.

The thalamic infarcts with pure sensory stroke reported thus far have been confined to the ventral posterior tier, in the regions traditionally considered to be the main sensory relay nuclei to the cerebrum. The lacunes in both of the reported autopsied patients have been quite small. If they are typical, it is easy to understand why so many thalamic lacunes thus far have escaped detection by CT scan.

The CT scan has been the basis of the other sites associated with pure sensory stroke. One case, thought to be due to a lacune because of its small size, affected the centrum semiovale, presumably with involvement of the thalamocortical projection area. Caution is necessary in this interpretation, as lacunes in the centrum semiovale are distinctly uncommon in series based on autopsy data. Involvement of subthalamic brainstem pathways has not yet been reported to be associated with pure sensory stroke. To my knowledge, the only autopsied patient with pure sensory stroke from a lesion outside the thalamus showed a small hemorrhage that involved the corona radiata of the posterior limb of the internal capsule.

The sensory complaints are often greatly in excess of the deficits found on clinical examination. In the most obvious instances, the disturbance in sensation characteristically affects one side of the body to the midline including genitalia, buttock, neck, and scalp, regions usually spared in hemispheric infarction. Striking alterations occur in spontaneous sensations: the parts feel stretched, hot, sunburned, pinpricked, larger, smaller, heavier. Skin contact from eyeglasses, bedclothes, rings, watches, or sheets feels heavy on the affected side and transiently aggravates the sensory disturbance.

The available literature does not support traditional claims that the face lies medially and the trunk and extremities lie laterally within the ventral posterior nucleus of the thalamus. In all cases reported with pathological confirmation, the disturbance affected both face and limbs, even though the infarcts were smaller than the confines of the ventral posterior nucleus.

Incomplete hemisensory syndromes are decidedly common, and the remarkable topography of the sensory complaints stretches notions of the homunculus to the limit. One autopsied patient suffered transient ischemic attacks affecting only the right fingers and, at another time, the right upper and lower lips, right side of the tongue, and the two medial toes of the right foot. At autopsy, a lacune 7 mm in diameter was found to affect the left ventral posterior nucleus. Others have involved the face, arm, and leg; head, cheek, lips, and hand; face, fingers, and foot; shoulder tip and lower jaw; distal forearm alone; fingers alone; and leg alone. Lapresle and Haguenau found partial sensory syndromes involving the face, the arms, the leg, the oral cavity, the peribuccal area and forearm, and the peribuccal area and radial edge of forearm, all due to focal thalamic softenings of lacunar size.

Some syndromes may be delayed in onset. The Dejerine-Roussy syndrome originally was described as the effect of occlusion of the thalamogeniculate branch of the posterior cerebral artery, with infarction of the ventral posterolateral and ventral posteromedial nuclei, largely sparing the remaining nuclei of the thalamus. Cases documented only by CT scanning have shown a lesion small enough to qualify for a clinical diagnosis of lacunar infarction. In these instances, the initial deficit usually includes a hemiparesis and hemisensory syndrome. The pain, which is an constant feature in patients with such infarcts, may begin at the onset of the syndrome or appear only later. Delays up to several months are common. Pains are intermittent or constant, appear spontaneously or are provoked by contact with the affected parts. They usually are accompanied by many other disturbances in sensation, including tingling, feelings of excessive weight, and feelings of cold, although a few patients have been described in whom sensory function is normal to clinical testing. The special disturbance known as hyperpathia is particularly characteristic but not common; following a sensory stimulus, a disagreeable response occurs that is usually delayed in onset, may spread over a large area, persists after removal of the stimulus, and may even increase in intensity over sev-
eral seconds. The syndrome may outlast other features of the original stroke syndrome and even become permanent. No reliable treatment has been devised.

Associated disturbances in motor function, language, and vision might be expected in a setting of thalamic infarction but have thus far been unreported, save for that in a single example of sensorimotor stroke due to thalamic lacunae. Given the anatomy of the thalamus and its widely varying projections to the cerebrum, such syndromes should be encountered but have thus far eluded the most careful efforts of vascular neurologists in diagnosis.

Improvement appears to be the rule: the patient's condition often returns to normal within weeks. The topography of the shrinking deficit may be rather unusual. Improvement in the trunk with persistence in the distal extremities, common in hemispheral disease, is encountered only occasionally. In one patient, the deficit shrank to a vertical band from the axilla down the lateral trunk to the thigh, a finding encountered personally in several patients studied in southern Alabama.

Sensorimotor stroke has been documented in three autopsied patients. Although casually assumed to be the cause of many cases of sensorimotor stroke uncomplicated by disturbances in vision, speech, or language, lacunes are actually rare. Garcin and Larpesle appear to have published the first case report as part of a review of sensory disorders from thalamic infarction. Their patient was a 65-year-old woman in whom left hemiparesis and a combination of hypesthesia and dysesthesia in the left peribucal area and forearm developed suddenly. At autopsy, a small infarct was found straddling the intersection of the ventral posterior lateral and medial nucleus of the right thalamus. Involvement of the internal capsule was not mentioned. The second patient arrived at the emergency ward of this institution in the early phase of his syndrome. The sensory component preceded the motor by several hours. The syndrome evolved smoothly and steadily over approximately a day, then stabilized for many days before improvement began. The sensory component involved the entire half of the body, including the neck, ear, and genitalia. At autopsy months later, a well-developed 4 × 4 × 2-mm lacune was found in the ventral lateral nucleus of the thalamus with pallor of the adjacent internal capsule. The third patient’s illness began as a sensorimotor stroke, but he had "loss of all sensory modalities." Autopsy revealed a 3 × 3 × 10-mm infarct involving the ventral posterolateral nucleus of the right thalamus and adjacent internal capsule. No mention was made of the arterial anatomy of the lesion.

At least 11 clinical examples of sensorimotor stroke have been documented by CT scan. In these cases, the lesions have been fairly large. Dysphasia has been encountered by Daniel Hier and his associates (personal communication) in one patient with a small thalamic infarction documented by CT scan. The size of the infarct was large enough to include the ventral anterior and rostral ventral lateral nucleus, which might be too large for an infarct from primary disease of the thalamoperforant vessels. My colleagues and I have uncovered three examples of an amnestic state on high field 1.5 T MRI from lesions assumed to be infarctions affecting the anterior thalamic nucleus. Although these lesions were small in size like the classic lacune, the exact mechanism of such infarcts has not been established. Much work remains to be done.

**Hypertension**

A few comments on the patient’s hypertension seem warranted before reviewing the neurological features. Calcium oxalate stones with episodes of renal colic, the earliest at the age of 31 years, raise the possibility of primary hyperoxaluria or of secondary hyperoxaluria attributed to fat malabsorption or thiamine deficiency. The possible ensuing renal failure may be a cause of hypertension. Since none of these diagnoses are obvious from the protocol, I would suspect he is one of the idiopathic stone formers. Given his tendency to form calcium stones, the thiazide diuretic given on his first hospitalization seems a good choice, especially in view of the prompt blood pressure response. What advantages accrued from substituting propranolol after his last admission are not clear to me.

Assuming the thalamic lesion represents infarction, and noting his modest hypertension, it is tempting to suggest another uncommon diagnosis, that of premature arterial disease associated with the heterozygote state for homocystinuria. This condition is said to occur in a frequency of 1:70 to 1:200 in the normal population and is associated with arteriosclerotic lesions of the peripheral and cerebral vessels. Presumably, a deep infarct from microatheroma could also occur. Apparently, the patient did not have an oral loading test of L-methionine, 0.1 g/kg body weight, in 1984; however, has he had it since then?

**Differential Diagnosis**

The differential diagnosis in this patient presents several problems. First, there’s the issue of the patient’s private life. If it weren’t for the possibility of acquired immune deficiency syndrome (AIDS), the reference to the patient’s sexual preferences might not be deemed necessary. His claim of monogamy might be inferred to suggest that he is safe from AIDS, as does his continued good health up to 8 months later. But the 8 months ended in 1984. Is he still healthy? Exposed as we are in New York to more cases of AIDS than we seek, we have been dismayed at the variety of neurological presentations of this syndrome. As familiar as we are with infectious, neoplastic, and paraneoplastic syndromes associated with AIDS, none thus far have been from infarction. To date, infarction attributed to arteritis with herpes zoster ophthalmicus and hemorrhage from autoimmune thrombocytopenia are the cerebrovascular syndromes associated with AIDS. The CT scans in AIDS cases may show a small focus of low density attributed to infarction, but
the cause usually has not been determined. I have heard elsewhere that such a patient has been autopsied here recently. Could this be the man? If so, I have no reason to make the diagnosis of AIDS with confidence. The laboratory study results, often abnormal in AIDS, were normal in this patient, and the normal rheumatoid factor, antinuclear antibody, and complement levels argue against the other major disorders associated with arteritis.

One final arteritis diagnosis, that of granulomatous arteritis confined to the central nervous system, has such a wide range of presenting signs and spinal fluid findings that its mention is worthwhile in almost any instance of unusual focal and nonfocal presentation of neurological syndromes.

The patient’s past history leaves me uneasy as well. The severe headaches with vomiting could be migrainous in character, although the early age of onset (5 years) is unusual. Although it is popular to think of arteriovenous malformations with headaches beginning at an early age, the syndrome discussed herein has not yet been part of the arteriovenous malformation picture in our experience in New York. As for the “unusual difficulty in controlling movements of the left leg” and the observation that “his gait deteriorated dramatically at that time,” more details are needed before these findings can be placed in perspective.

It might be argued he had a herniated disk, which could easily have been aggravated by sneezing; however, the clumsiness of the left hand would not be explained by this diagnosis. Although multiple sclerosis is a diagnosis to be considered in a young man who has had more than one episode and a different syndrome with each, his spinal fluid was clear and the visual evoked potentials were reportedly normal.

The concern for the left-sided motor complaints might be deemed an unnecessary distraction, except that a few examples of thalamic lacune exist, documented by autopsy or image technology, that began as a movement disorder. One started as a sensorimotor stroke, in which hemiatrophy developed after some weeks. In such patients, we have been conspicuously unsuccessful in imaging such tiny lesions in life, even using our own high field 1.5 T MRI unit. Thus, there is the remote possibility that the lesion seen on MRI scan (I believe a 0.5 T MRI unit was used) could be a sign of his left-sided motor disturbance of 1982 and not the present lesion.

The complaints of “jumbled vision,” if translatable to diplopia from skew, might mean that the basic disease involved vessels larger than the tiny twigs usually involved in producing lacunar infarction and should call for a magnification angiogram of the top of the basilar artery. The protocol suggests that the conventional angiogram was normal.

The December 1984 episode deserves comment. The sensory symptoms began in the face and spread within seconds to the arm and leg. Because involvement of the scalp, neck, trunk, and genitalia is such a helpful sign of thalamic involvement, it would be worthwhile to know whether the patient was spared such involvement or whether the protocol has merely been spared such details. The point is of especial interest since he is described as having difficulty controlling the leg, a sign that might mean the lesion affects not only the ventroposterior nuclear region but the adjacent ventrolateral nucleus as well. Such a combination of lesions should mean one larger than the usual lacune, especially if the sensory loss was so circumscripted. Similarly, it is interesting that the sensory loss faded so quickly from the face — the region first involved — then gradually from the arm and from the leg, the limb affected by the movement disorder. Since no further mention is made of the movement disorder, perhaps this difficulty in controlling the leg was nothing more than faulty placement from sensory loss.

That his lesion could be seen on an MRI unit of 0.5 T and that his sensory evoked potential was deranged suggest a lesion of considerable size. Visible lesions on CT scan usually have been associated with larger lesions producing a combination of sensorimotor weakness. Those visualized by low field MRI units usually have been associated with persistent and complete hemisensory loss. Deranged somatosensory evoked potentials have also been a sign of larger lesions. Yet the present patient’s sensory deficit is said to have faded by 72 hours. We have no such example in our series of pure sensory stroke or sensorimotor stroke. The closest was one seen by a colleague in Alabama, but the patient had a persistent hemisensory syndrome that also involved the trunk, as would be expected.

The contrast of negative clinical and positive laboratory findings in the present patient keeps me suspicious that there is more to this case than the usual thalamic lacune. If his condition is due to a single lacune, it is almost certainly a unique occurrence.

Questions and Answers

Dr. William H. Sweet (Massachusetts General Hospital, Boston, Massachusetts): How frequently does a patient complain of such pronounced sensory loss when results of all sensory evaluations are normal?

Dr. Mohr: I think it is almost the rule. I would agree with Dr. Fisher’s point, which he made in Stroke a year or so ago, that subjective sensory complaints are extremely common. Those cases we label as lacunar stroke resulting in pure sensory stroke and having findings on examination are infrequent, but subjective complaints in the absence of an obvious finding on examination are, I think, extremely common. This case is exceptional, I think, and the first one with which I am familiar in which both a lesion on MRI scan and an abnormality in the sensory evoked potential are reportedly present in the absence of persisting sensory symptoms. This case is without precedence in my experience.

Dr. Sweet: Wouldn’t he have had graphesthesia or loss of two-point discrimination, apart from the localization of the distinction tests?
Dr. Mohr: I would assume, since Dr. Stakes saw him, evidence for such objective loss of sensation was evaluated many times over.

Dr. John Stakes (Massachusetts General Hospital, Boston, Massachusetts): A point to be raised concerns the rather subtle abnormalities of the median and tibial somatosensory in this patient. In fact, the abnormalities on median nerve stimulation were really limited to an asymmetry in the amplitude of that N19 waveform. In the tibial somatosensory, it was an asymmetry in the sort of interwave latency differences outside the range of normal that suggested a disturbance in conduction. Despite that knowledge and the subsequent findings on the MRI scan, a marked abnormality was never detected in tests of sensory function. The patient, parenthetically, is a psychologist and very introspective and helpful in providing the clinical history. One other point should be added to the historical information. In the spring of 1982, he described difficulty in moving his left leg. He said he walked with the leg rather stiffly and in an arc, and it seemed to be beyond his voluntary control for some time. He was troubled by the fact that his walking had taken on an involuntary aspect and he had to be concerned about leg placement at that time.

Dr. Mohr: So it is not unreasonable to suspect that the 1982 lesion could be what we are seeing on the MRI scan now.

Dr. Stakes: Yes, though there were no sensory complaints at that time.

Dr. David N. Levine (Massachusetts General Hospital, Boston, Massachusetts): Was the posterior cerebral artery seen angiographically?

Dr. Mohr: I infer that it was, from the protocol, and I also infer that the visible branches at the top of the basilar artery were also considered to be clean.

Dr. Robert M. Graham (Massachusetts General Hospital, Boston, Massachusetts): What intrigued me about this patient was the relatively mild severity of his hypertension. I see a lot of hypertensive patients, and this is the first one I have treated that has had a stroke with this degree of mild blood pressure elevation.

Dr. Mohr: That is one of the points that concerned me in casually accepting the diagnosis of thalamic lacune. One of the principles of establishing a diagnosis of lacune, as opposed to an infarct from other forms of arterial disease, is the hope that this is a finished process and that control of hypertension might delay the day when other small or large arteries get into trouble, when a biopsy is needed, or when invasive procedures like angiography, which in some institutions has as much as a 4% incidence of complications, are required.

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